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APPENDIX I

ANALYTICAL PLAN FOR AIR TOXICS CASE STUDY - BENZENE EMISSIONS REDUCTIONS IN HOUSTON

PURPOSE AND SCOPE OF THE CASE STUDY

The purpose of this document is to refine the analytical plan for a hazardous air pollutant (HAP) benefits assessment to accompany the main criteria pollutant analysis in the second 812 prospective study. Efforts to characterize the benefits of HAP reductions under Title III in prior 812 analyses have been only partially successful. An analysis of NESHAP regulations conducted for the retrospective analysis was criticized by the SAB as substantially overstating benefits, with particular note made of the use of "upper bound" dose-response relationships (i.e., the cancer potency factor used for standard setting). EPA made a second attempt to incorporate air toxics benefits, in the first prospective analysis, but the SAB felt the national air quality and exposure model proposed (ASPEN/HAPEM) would not yield estimates suitable for benefits analysis. In July 2001, however the SAB Council proposed that EPA undertake a case study, and suggested benzene as a good candidate pollutant. This document focuses on the development of a case study of the benefits of benzene emissions reductions attributable to CAAA regulations.

In the original analytical plan, we proposed to estimate only the VOC benefits of HAP controls, as part of the larger criteria pollutant analysis. Building off the results of the then-recent SAB-triggered workshop on air toxics benefits analysis, we concluded that the available tools were not appropriate for a comprehensive benefits analysis. We further proposed to conduct cost-effectiveness calculations (cost per ton HAP reduced).

In response to the original analytical plan, the SAB issued the following comments:

! **Representative HAP analysis.** The SAB advises the EPA to work with the National Air Toxics Assessment to select one representative Hazardous Air Pollutant (HAP) for which to perform a prototype 812 analysis. The SAB recommends benzene because of the wealth of available national ambient concentration data, but notes that toxic metals such as arsenic and cadmium are also options.

! **Benzene as prototype.** The SAB feels that an 812 analysis using the available benzene data would:

- identify limitations and gaps in the data base,
- provide an estimate of the uncertainties in the analyses and perhaps provide a reasonable lower bound on potential health benefits from control, and

- provide a scientific basis for deciding whether there is merit in pursuing a greater ability to assess the benefits of air toxics.

! **AQM and exposure analysis.** The SAB questions whether the sort of national modeling that is being performed for ozone and nitrogen (PM) assessments is appropriate for benzene.

In response to these comments, we are proposing to undertake a metropolitan scale analysis of the benefits of Clean Air Act controls on benzene emissions. The smaller scale will allow us both to perform a more rigorous analytical effort and to build on previous EPA modeling efforts for benzene. We propose a local-scale study of the Houston, Texas area (Harris county, specifically); this approach will allow EPA to utilize existing modeling data developed for an ongoing air toxics study in this area. The analysis will be designed to capture benefits of reductions in benzene resulting from multiple CAA Titles and provisions.

While the focus of the 812 analysis of HAP benefits remains the benzene case study recommended by the SAB, EPA has also been making progress in recent years addressing the SAB Council's earlier concerns about the data and modeling tools available to support national-scale assessments of benefits in previous 812 studies. Therefore, EPA plans to explore the feasibility and appropriateness of conducting a national-scale analysis to supplement the case study approach planned for the current 812 study. If such a national-scale assessment is conducted, advice pertaining to the merits and design of such an assessment will be sought during a future SAB review.

The analytical framework for this analysis will follow the approach for benefits analysis used in the criteria pollutant analysis of the Section 812 study. The framework includes the following steps: Scenario Development, Emissions Estimation, Air Quality Modeling, Exposure Assessment, Health Effects Estimation, and Benefit Valuation. Our plans for these steps are described in detail in the following sections.

SCENARIO DEVELOPMENT

As in the criteria pollutant analysis, the HAP case study relies on detailed descriptions of the pre-CAAA and post-CAAA scenarios. We propose to define reasonable scenarios describing benzene emissions control requirements as currently implemented and as they would be in the absence of the CAAA. The differences in the emissions, impacts, and benefits realized under these two scenarios represent the primary results of the analysis.

We define the scenarios to be consistent with those in the criteria pollutant analysis. That is, the pre-CAAA scenario freezes Federal, State, and local benzene controls applicable to Houston at 1990 levels, and the post-CAAA scenario includes all Federal, State, and local benzene rules enacted in response to the 1990 CAAA. However, due to resource considerations, we are proposing to limit the study period for the HAP case study to 20 years, from 1990 to 2010.

Although this is a case study of a hazardous air pollutant, we do not propose to analyze benefits specific to Title III of the CAAA (the Title that specifically focuses on HAPs), because doing so would ignore significant benefits related to reductions of benzene emissions from mobile and stationary sources. Instead, the difference between the pre- and post-CAAA scenarios for benzene in Houston will reflect the effect of all CAAA regulations that affect benzene emissions.

Pre-CAAA (Baseline) Scenario

This scenario will be consistent with the baseline scenario for the main analysis. It will assume no further controls on benzene emissions beyond what was in place in 1990, prior to issuance of the amended Clean Air Act. Details of the regulations included in the pre-CAAA scenario can be found in Chapter 2 of the 812 Analytical Blueprint.

Post-CAAA (Control) Scenario

This scenario will include all current and currently anticipated regulations that affect benzene emissions resulting from the amended Clean Air Act issued in 1990. We expect the scenario will include the regulations listed in Exhibit I-1.

Exhibit I-1 **Benzene Case Study Post-CAAA Projection Scenario Summary, by Title**	
Title I	Any effects of Title I will be expressed through state implementation plan (SIP) requirements, such as (enhanced) I/M programs, transportation control measures, other VOC controls. These requirements are dependent on the ozone non-attainment status of the case study area(s).
Title II	**Tailpipe standards** **Onroad** Tier 1 Standards (phased in 1994 to 1997) NLEV program –voluntary bridge between Tier 1 and Tier 2 Tier 2 Standards take effect in 2004 Heavy Duty Engine/Diesel Fuel Rule - New emission standards – 2007 model year, new fuel standards 2006 **Nonroad** Federal Phase I and II compression ignition (CI) engine standards, Federal Phase I and II spark ignition (SI) engine standards, Federal locomotive standards, Federal commercial marine vessel standards, Federal recreational marine vessel standards. **Evaporative Emissions** Stage II Vapor Recovery Systems (Section 182) Onboard Refueling Vapor Recovery (Section 202; 1998 model year and on) Fuel Spit-back rule Clean Fuel Vehicle Program **Fuel Regulations** RFG Standards (1995 on) Phase II – (2000 – present) – benzene requirements essentially unchanged Anti-dumping standards – do not specifically regulate benzene content of gasoline Summertime Volatility Requirements for Gasoline (Phase II – 1992 on) Anti-backsliding provisions of Mobile Source Air Toxics rule
Title III	**MACT Standards** We will review the full range of MACT standards to identify those that would be expected to have a significant effect on future-year benzene emissions in the Houston area. We expect that the final list of MACT standards to be analyzed in the study will include: Oil and Natural Gas Production: 7-Year MACT Petroleum Refineries: 4-Year MACT Gasoline Distribution: 4-Year MACT Pulp and Paper Production: 7-year MACT Municipal Landfills: 10-year MACT Natural Gas Transmission and Storage: 10-year MACT Publicly Owned Treatment Works (POTW) Emissions: 7-year MACT Coke Ovens: Pushing, Quenching, & Battery Stacks: 4-year MACT Synthetic Organic Chemical Manufacturing: 2-year MACT

EMISSION ESTIMATION

This section provides a brief overview of our approach to developing emissions inventories for benzene for use in the HAP case studies to be included in the second 812 prospective analysis.

To facilitate this analysis, we seek to build on previous emissions estimation efforts by EPA, while still maintaining consistency with emissions estimation for the main 812 analysis.

Available Emissions Data Sources for Benzene

The primary data source for benzene (and other HAP) emission estimates is the National Toxics Inventory (NTI) which has recently been renamed as the National Emission Inventory for Hazardous Air Pollutants (NEI for HAPs). EPA's Office of Air Quality Planning and Standards (OAQPS) is using the NEI for HAPs to support analyses required by the Clean Air Act and Government Performance and Results Act (GPRA) that depend on a high quality, comprehensive HAP emission inventory. The inventory is a critical component of the entire national air toxics program. A recent example of its use is in the EPA National Air Toxics Assessment (NATA).

The NEI for HAPs is developed every three years (1993, 1996, 1999, etc.) with the draft version 3 of the 1999 NEI for HAPs being the most recently completed version. The final version 3 of the 1999 NEI for HAPs is expected to be completed in July. The NEI for HAPs contains emission estimates for large stationary sources (point), small stationary sources (non-point), and mobile sources. Point sources in the inventory include major and area source categories as defined in Section 112 of the Clean Air Act. Non-point source categories in the inventory include area sources that are not included in the point sources and other stationary source categories. Individual emission estimates are developed for point sources, while aggregate emission estimates at the county level are made for non-point stationary and mobile sources. For all inventory years, the NEI for HAPs also identifies emission sources that are associated with MACT categories.

In addition to the NEI for HAPs data years, the benzene analysis approach also considers recently completed/ongoing HAP studies performed for the Houston-Galveston, Portland (Oregon), and Philadelphia areas.

Recent EPA Efforts to Improve Emissions Projections

EPA's Office of Air and Radiation has participated in three urban scale studies of air toxic emissions and associated concentrations, which are at different stages of completion. The three urban areas are Houston, Portland (Oregon), and Philadelphia. All three studies examined benzene as one of the HAPs evaluated and employed on-road emission estimation methods that involve some improvements to standard methods like allocations of emissions to major roadway segments. Two of the three (Houston and Philadelphia) use the ISCST3 Gaussian dispersion model to estimate ambient benzene concentrations, while the CALPUFF model is used in the Portland study, where terrain effects are more of a concern.

EPA's OAR completed urban scale modeling analyses and evaluations in Houston using 1996 HAP emission estimates, with benzene being one of the four HAPs included in the analysis. The Houston domain for the EPA study included all of the Houston-Galveston-Brazoria ozone nonattainment area counties, which are Brazoria, Chambers, Fort Bend, Galveston, Harris, Liberty, Montgomery, and Waller.

The Portland Air Toxics Assessment is a pilot project funded by EPA in cooperation with the Oregon Department of Environmental Quality. The reference material available for the Portland study describes the on-road vehicle air toxic emission estimation procedures used to calculate hourly air toxic emissions by roadway link and travel analysis zone for the Portland-Vancouver area.

The third EPA urban study of air toxics is currently being performed for the Philadelphia ozone nonattainment area. The counties in the Philadelphia urban study domain include five Pennsylvania counties, five New Jersey counties, and one county in Delaware. The base year for this study is 1996. Study documentation available to date primarily addresses emissions processing steps. The processing steps involve running EMS-HAP programs and a post-processing program designed to split the domain into rural and urban portions, so that the air dispersion model – ISCST3 – is applied separately for urban and rural domain emissions. Benzene is one of the nine HAPs evaluated in the Philadelphia urban study.

While it may be desirable to pursue benzene analyses for all three potential urban areas of interest, this proposed analytic approach focuses on the data and analysis tools available for the Houston area that would be used to perform the needed evaluations for the second prospective. Techniques that might be applied to Portland, Oregon, or Philadelphia in the 812 assessment would be expected to be similar (but not exactly the same).

Necessary Modifications to Ensure Consistency with Main 812 Analysis

The tool that has been developed by EPA-OAQPS for performing HAP emission projections is EMS-HAP. This model has the ability to employ the same EGAS 4.0 growth factors that we propose to use in the criteria pollutant analysis in order to account for likely changes in pollution generating activity in future years affecting HAP sources. For deployment in the 812 analysis, there will need to be adjustments made to start the projections with 1999 base year emissions data and to estimate future year emissions in 2010.

Note that the growth factors in EGAS 4.0 for the Houston area are developed from a Regional Economic Models Inc. (REMI) regional model that distinguishes the Houston area from other urban/non-urban areas in Texas. However, because some source categories' (e.g., fuel combustion) growth factors are based on forecasts from non-REMI sources (e.g., Department of Energy), there will be source categories where EGAS will have the same growth factor for Houston as it does for the entire State.

Proposed Approach by Source Type

This section presents our proposed approach to benzene emissions estimation (including base inventory source, any necessary adjustments, and projection methods) for each of the major source categories we plan to include in the analysis.

Point Source Emissions

EPA has modeling inventories for Houston point sources for 1990 and 1999. The 1990 point source inventory was prepared by the Texas Commission on Environmental Quality (formerly the TNRCC) for Harris County. This data set is considered the best estimate of point source benzene emissions in 1990.

Similarly, the 1999 point source benzene emission estimates in the 1999 National Toxics Inventory, version 3 final, are the recommended data source for estimating recent (1999) emission levels. This version is expected to be completed in July 2003. With the significant reductions in reported air toxic emissions between 1990 and 1999, the suggested 1990 and 1999 point source data bases should provide the best indicator of post-CAAA scenario emission changes in this time period. Post-CAAA scenario benzene emissions for 2000 in Houston can be estimated by either using 1999 values as a surrogate, or performing a one-year projection from 1999 to 2000.

One of the key parts of this analysis will be identifying the point source benzene emission reductions attributable to MACT standards promulgated during the 1990s. To evaluate EPA's progress in reducing air toxic emissions via MACT standards, and to identify sources that may be modeled as part of residual risk assessments, operations within facilities that are subject to MACT standards are identified in the NEI for HAPs by MACT codes. MACT codes are assigned at the process level, or at the site level. For example, the MACT code for municipal waste combustors is assigned at the site level, while the MACT code for petroleum refining catalytic cracking is assigned at the process level. These MACT codes are expected to be used as an indicator of where MACT standard associated emission reductions have occurred (by 1999) or are likely to occur in future years.

One of the most important issues in the 2010 emission projections for the Houston case study is determining the appropriate level-of-detail for evaluating the expected benzene emission reductions to attribute to the CAAA measures post-1999. One way to do this is to survey the MACT-standard affected facilities in order to determine their compliance plans. However, such an effort would likely be resource intensive and time consuming. In addition, it is not clear what authority EPA has to survey the Houston area facilities in order to gather the data needed to accomplish this approach.[1] Another possibility is to work with the state agency to see if they have

[1]Paperwork Reduction Act compliance requires a rigorous application process for any survey effort involving more than nine participants.

this level of information. Again, such an effort would be resource intensive and time consuming and may not yield any of this specific data.

Our proposed approach to developing the post-CAAA scenario benzene point source emission estimates will include estimates of the likely emission reductions by facility, or source category, needed to meet residual risk requirements of Title III, if these data are available within the time frame of our analysis.

- **Option 1:** EMS-HAP contains future year control factors by source category that are designed to be applied to 1996 base year emission estimates to include the effects of MACT standards implemented post-1996. These control factors capture the average VOC HAP emission reduction expected when the new standard is applied to all affected sources in the country. Thus, the estimated nationwide emission reduction associated with a MACT standard might be 45 percent, when the range of emission reductions by individual sources varies from 0 (facilities on which the MACT standard is based) to 90 percent (at a previously uncontrolled facility). Applying the 45 percent control factor in Houston could greatly under-or-over estimate the MACT standard benefits for a source category.

- **Option 2:** The analysis alternative (for developing control factors) that is most like the criteria pollutant approach is to develop an estimate of the VOC/benzene emission control efficiency required by the applicable MACT standard for each affected source category, and to then compare the existing (1999) VOC/benzene control efficiency with that MACT standard control level to determine whether the facility is expected to be adding controls in order to meet the MACT standard requirements. This approach for modeling the future year benzene emission reductions associated with each point source category in the Houston area can only be applied if base year control efficiencies are available for MACT standard category affected units. If they are, then the MACT standard requirement can be compared with the existing control efficiency, and a further emission reduction applied if the existing control efficiency is less than what is required by the MACT standard. EMS-HAP has the capability of accounting for base year control efficiencies in computing the actual expected control efficiency with a future MACT standard. However, the base year efficiency must be in the input inventory.

  It is also our understanding that the Houston ozone SIP has been recently revised to include additional point source VOC emission controls, and that these measures may have some effect on benzene emissions at the affected facilities. Thus, control factors for the chemical and petroleum industry sources in the Houston area would have to be assembled from SIP documents and an analysis of MACT standard effects on these same sources.

- **Option 3:** Another point source analysis alternative is to see whether the EPA MACT standard Background Information Documents contain enough information to characterize the before and after MACT standard performance of the particular (either as a whole or individually) chemical and petroleum industry facilities in the area.

- **Option 4:** The simplest point source analysis alternative is to assume that the 1999 point source file emission estimates capture the majority of the post-CAAA emission benefits, and that benzene emissions will be relatively constant thereafter. This overlooks the benefits of 7- and 10-year MACT standards in the area and the recent Houston-Galveston area SIP requirements that are expected to further reduce point source VOC emissions.

The pre-CAAA scenario 2000 and 2010 point source emission estimates will be generated by applying expected increases in activity levels assuming no additional controls are implemented beyond those that were in place in 1990. Activity changes will be estimated by applying EGAS 4.0 growth factors for the Houston-Galveston area by SIC or SCC code for the 1990 to 2000 and 1990 to 2010 periods.

Given the information available, our primary recommendation is that the HAP Case Study analysis be limited to the benzene emission sources in Harris County, Texas. This allows us to focus our efforts on quantifying the estimated effects of the 1990 CAAAs on point source benzene emitters between 1990 and 1999, and the likely future changes post-1999. From a modeling standpoint, because the transport of benzene emissions from other nearby counties in the urbanized area will not be captured, it will be important to set appropriate background concentration levels to capture this contribution to ambient benzene levels in Harris County.

If the benzene analysis is performed for all eight counties in the Houston-Galveston ozone nonattainment area, then there will be significant additional effort needed to prepare point source benzene emission estimates for 1990. This would involve taking the 1999 point source file for these counties and backcasting these estimates to 1990 conditions. The primary approach that would be used to prepare 1990 benzene emission estimates for these point sources is to use data collected by EPA's Emission Standards Division during the MACT standard-setting process to estimate pre-MACT standard operating conditions and emissions.

Highway Vehicle Emissions (On-Road)

The benzene emission factors used in the 1996 Houston study were from MOBTOX (the predecessor to MOBILE6). MOBTOX-estimated benzene emission factors were estimated using a 19.6 mile per hour average speed and standard Federal Test Procedure cycle hot and cold start percentages. MOBILE6 and MOBTOX fuel parameters are the same.

In the Houston analysis, on-road emissions are modeled in ISCST3 in two ways. The first was to assign on-road emissions to 1 kilometer (km) grid cells. A second method was to allocate on-road emissions to major road segments such as Interstate, U.S., and State highways using GIS software. On-road vehicle emissions not specifically allocated to these roads were interpolated to 1 km grid cells. Therefore, for the 812 analysis, either of these two options could be used to estimate on-road benzene emissions.

- **Option 1:** Use county-level on-road benzene emission estimates and allocate to 1 km grid cells.

- **Option 2:** Where possible, place roadway emissions at actual locations using a GIS system and activity estimates for individual links (vehicle counts).

EPA prefers Option 2 because the dispersion model performance is better in Houston when this option is employed.

It is proposed that the on-road ISCST3 input file for 1996 will be used as the base file for the analysis, with scaling factors applied to these 1996 benzene emission estimates to estimate on-road vehicle benzene emissions for all of the Section 812 analysis years of interest. The scaling factors will account for MOBILE6-estimated emission factors and expected vehicle miles traveled (VMT) changes in each analysis year. It is expected that VMT projections for Harris County will be available from the Houston-Galveston Area Council to support our ability to estimate likely future year VMT changes by year and geographic area within Harris County. The proposed approach is to use available travel demand model projections for the area to prepare estimates of 2010 and 2020 VMT at the 1x1 km grid cell level. Because Houston's attainment year is 2007, it is expected that VMT projections will be available for that year. In addition, the area will have also had to prepare a long-range forecast for transportation conformity purposes. Their current efforts are in preparing a long range forecast to 2030. A year closer to 2020 may have been included in previous transportation conformity analyses. In any event, it is expected that some interpolations will be required to incorporate Harris County-specific VMT forecasts in the 2000 and 2010 HAP analysis. Our objective is to capture the expected changes in traffic patterns across Harris County in future years. Because traffic counts were used to estimate base year VMT by geographic area, and future year VMT estimates are expected to come from the travel demand model, there will have to be some reconciliation of travel demand model-estimated traffic county-estimated VMT, as well.

One of the factors in how we approach revising the on-road benzene emission estimates to incorporate MOBILE6 and to model different scenario years is the complexity associated with separating the different exhaust and evaporative benzene components, and allocating these spatially/temporally. Benzene *all vehicle* MOBILE6 emission factors for average Texas conditions are shown below:

Emission Factor Components	Benzene mg/mile
Exhaust	58.18
Hot Soak	1.54
Diurnal	0.32
Resting Loss	0.58
Running Loss	5.14
Total	66.03

In the Houston analysis, MOBTOX was used to estimate on-road benzene emissions, and we now want to use MOBILE6 to generate on-road emission factors. For calendar year 1999, the *national* on-road benzene emissions are estimated to be 174,720 tons per year using MOBILE6, and were estimated to be 165,700 tons per year using MOBTOX. This is a 5 percent increase with MOBILE6. Because this is an annual estimate for the entire United States, there could be bigger differences in specific areas and seasons. While the Houston analysis will include fuel parameters and other conditions particular to that area, the national level benzene emission differences provide a sense of what the MOBTOX to MOBILE6 adjustment might be.

In case the Philadelphia or Portland areas are to be included in the 812 HAP case study, some information about how their on-road emissions analysis methods differ from those used in Houston is provided below. The information provided in this analytical plan outline for these two areas is limited to on-road vehicle emission estimation methods because this source type was a point of emphasis in both studies.

The information available for Portland, Oregon focuses on on-road vehicle emission estimation methods. Methods applied to estimate current year HAP emissions are more sophisticated than those used for the Houston area inventory. The primary methods improvements compared with those used in Houston include accounting for differences in vehicle speeds and their effect on emission rates, differentiating running and non-running emissions -- with allocations of non-running emissions to trip origins, and using household survey results and the trip assignment model for Portland Metro to allocate travel by hour of the day. Separate MOBILE6 runs were conducted for each combination of area *fleets*, two seasons, four link types, and 14 speed bins. Speed curve equations were generated to allow benzene emissions to be computed for any associated speed. MOBILE6 emission factors were applied at a link level to compute running emissions by hour. Emissions from intra zonal travel, and all non-running emissions, were allocated at trip origins.

If the analysis is extended to the Philadelphia area, the on-road vehicle emission estimation methods used in Philadelphia are consistent with those applied to estimate Portland area emissions.

For the pre-CAAA scenario, we need to estimate what the fuel parameters were likely to have been in the absence of regulation (using 1990 values is one option). The remainder of the MOBILE6 set-up for the pre-CAAA HAP scenario will be consistent with that being performed for the criteria pollutant analysis. These data are available from the procedures used for the NEI.

Non-road Vehicle and Engine Emissions

For the off-road sector, most source categories are included in EPA's NONROAD model, so the latest version of NONROAD will be the recommended model for estimating benzene emissions (where benzene will be estimated as a fraction of VOC emissions). Off-road vehicles/engines source types not included in NONROAD are aircraft, railroad locomotives, and marine vessels.

For the source categories in Houston whose benzene emissions for 1996 were estimated using the NONROAD model, for each analysis year, the most recent NONROAD model will be used to develop a benzene emission factor for each source category. The ratio of the new emission factor for each analysis year to the previously estimated 1996 Houston benzene emission factor will be used to develop a composite non-road benzene emissions scaling factor that will be used to adjust the gridded benzene emissions file (input to ISCST3).

For the 1996 Houston analysis, special processing was performed for aircraft emissions. These emissions are separated from the mobile inventory using Airport Proc. This program separates airport emissions from the mobile inventory and prepares the airport emissions for input into the point source processing programs. Airport Proc allows for modeling airport-related emissions as ISCST3-area sources with known locations and dimensions, rather than as spatially allocated mobile sources. This capability was built into the program because airport locations are known. EMS-HAP has been revised (though not yet documented) to include this capability for airport-related emissions from the area source inventory (e.g., aircraft refueling) and generalize it to include other traditionally nonroad or non-point sources in which specific locational data could be supplied.

For categories not included in NONROAD, we propose to apply growth and control factors (VOC factors) developed for the criteria pollutant analysis to develop consistent emission projections for benzene. Some adjustments may be necessary to aircraft emissions so that they can be processed separately for input to point source processing programs.

To configure the NONROAD model to remove the effects of the CAAA for the pre-CAAA scenarios, we expect to develop a specialty input file for NONROAD. This input file will be used to simulate emission rates if uncontrolled 1990 emission rates persist. All non-road engine emission standards are attributable to the CAAA, so we need uncontrolled 1990 emission factors to apply to the expected activity in each projection year.

Area Source Emissions (Non-Point)

For non-point (area) sources, the criteria pollutant analysis is designed to use the 1990 and 1999 National Emission Inventory emission estimates for most source categories to reflect the emission changes for the post-CAAA scenario. The exception to this is for source categories with significant emission estimation method changes in this period. The same basic approach is proposed for benzene. We need to determine which source categories have had the most significant methods changes. Fire emission estimates were mentioned in a recent conference call as one candidate for separate treatment.

Within non-point, one of the most prominent benzene sources is service station emissions. An important component of these emissions is vehicle refueling (because of the personal exposure). MOBILE6 is the preferred tool for producing emission factors for refueling because it can account for the combined effects of any Stage 2 controls plus the onboard vehicle refueling controls that have appeared on new gasoline-fueled vehicles since the mid-1990s. It is our understanding that the Houston ozone nonattainment area counties implemented Stage 2 controls in 1993. Therefore, the post-CAAA scenario benzene emission estimates for Houston will include these Stage 2 associated emission reductions (at a 95 percent control efficiency). The 1990 and pre-CAAA benzene emission estimates will be at pre-control (uncontrolled) levels. While service station emissions are typically represented in the non-point source data base, there may be service stations included in the point source data base for the area which will have to be reconciled with the non-point source estimates.

For the pre-CAAA scenario analysis, it will be necessary to identify the area source categories that emit benzene that have been affected by 1990 CAAA provisions. This may be a combination of Title I - Nonattainment provisions designed to reduce ozone precursors and Title III requirements. One way to investigate this is to identify where control factors have been applied in estimating benzene non-point source emissions in the 1999 NEI for HAPS.

> ***We need to determine which benzene-emitting area source categories have had significant methods changes between when the 1990 and 1999 area source emission estimates were produced. For these categories, 1990 benzene emissions will be re-calculated using methods consistent with those used for 1999 estimates. Because the Texas CEQ submitted its own 1990 area source emission estimates based on some specialized surveys performed for Harris County, and the 1999 draft benzene emission estimates include some non-point source submittals from the State, some effort will have to be spent ensuring that regulation-affected benzene emissions can be pinpointed.***

AIR QUALITY MODELING

The choice of an air quality model for use in a specific geographic area depends on several factors, including the complexities of weather and terrain in the area; the level of detail available in the emissions inventory, and the schedule and resources of the project. While many air quality models could be used to assess ambient concentrations of a HAP in an urban area, we have selected the Industrial Source Complex - Short Term (ISCST3) model for this analysis. ISCST3 is a steady-state Gaussian plume model used to assess pollutant impacts from multiple point, area, and mobile sources.

An air dispersion model used to estimate air toxic pollutant concentrations in an urban area should meet certain criteria. Ideally, the model should:

- be readily available;
- represent state-of-the-art modeling practice;
- be applicable to urban areas and irregular terrain;
- be capable of handling point, area and mobile sources;
- be capable of accounting for dry and wet deposition of pollutants;
- be capable of treating atmospheric chemical transformations - pollutant chemistry;
- be capable of accounting for pollutant emissions that vary by season and hour-of-day;
- be able to group source types for assessing impact;
- be capable of providing annual average concentration estimates (as well as shorter time averages);
- be computationally efficient; and
- demonstrate good performance when compared with observed concentrations. (U.S. EPA, 2002)

The ISCST-3 Gaussian plume model is widely used for estimating the impacts of non-reactive pollutants such as benzene because of its good performance against field measurements, and because it is computationally efficient relative to other types of models, such as grid and puff models. The lack of complex terrain in the Houston area also makes ISCST3 a good choice for this analysis; cities with less level terrain may benefit from more complex models such as CALPUFF. Other features of the ISCST3 dispersion model that make it useful for modeling air toxics in an urban environment also include:

- modeling of multiple point, area, and mobile sources;
- incorporation of building downwash effects;
- availability of an urban dispersion option;
- flexibility in specifying receptor locations and grouping of source impacts;
- algorithms for assessing the effects of elevated and/or complex terrain;
- modeling of the effects of deposition of gaseous and particulate emissions;
- an option to vary emissions by season and hour-of-day; and

- an option to treat atmospheric transformations by exponential decay (U.S. EPA, 2002).

Additional details about the ISCST3 dispersion model can be found in the ISC3 model user's guide (U.S. EPA, 1995).

We plan to run ISCST3 for the base year, 1990, and each of the target years for both the pre- and post-CAAA scenarios to calculate annual average benzene concentrations at the population weighted centroid of each census tract in Harris County. We plan to use a similar modeling approach to that used by EPA in its previous analysis of benzene concentrations in the Houston area in 1996 (U.S. EPA, 2002)

Alternatively, if the source locations used in the 1996 emissions year modeling analysis for Houston closely correspond with the 1990 and 1999 source locations, a source-receptor approach to air quality modeling may be possible. If the source locations match, the most efficient way to provide model-estimated benzene concentrations for the pre- and post-CAAA scenarios is to estimate source-receptor coefficients (grids versus 701 receptors) and to use the grid-level changes in benzene emissions and the source-receptor coefficients to estimate benzene concentrations at each of the receptors for each scenario. The choice between ISCST3 runs and a source-receptor approach depends on how computationally efficient the ISCST3 model is. If the model set-up and computational time is minimal, then making additional model simulations would be preferred to developing the source-receptor coefficients.

We plan to evaluate the validity of modeled annual average benzene concentrations against monitoring data for benzene in the Houston area. Air toxics monitoring data will be obtained from EPA's Aerometric Information Retrieval System (AIRS) web site at http://www.epa.gov/airs. Agreement between modeled and observed values within a factor of two will be deemed acceptable for use in the modeling effort.

HUMAN HEALTH EFFECTS ESTIMATION

This section presents our proposed approach to estimating avoided adverse health effects in humans resulting from reductions in exposures to benzene in ambient air and in various microenvironments. We begin by describing how we translate the ambient benzene concentrations output from the air quality model into estimates of benzene exposures to individuals as they carry out their daily activities. We then explain how we calculate numbers of cases of avoided cancer cases due to changes in exposure levels, using dose-response data for benzene.

Exposure Estimation

We plan to estimate time-weighted average exposure concentrations to benzene for the general populations in the study area of interest, based on the output of the air quality models. In

addition, we plan to assess risk reductions to two specific high-exposure subpopulations: individuals living in homes with attached garages who spend most of their day at home, and service station workers. We plan to generate these estimates using the same exposure model used for the general population.

The options for exposure modeling for the HAP case study include 1) the latest version of Hazardous Air Pollutant Exposure Model (HAPEM), HAPEM5, and 2) the new Air Pollutants Exposure Model (APEX).

HAPEM was developed for use in the 1996 National Air Toxics Assessment (NATA) which attempted to characterize exposures and risks from high-priority urban air toxics for population groups nationwide, using available EPA toxicity data. The HAPEM model inputs the ambient air concentrations from an air quality model, and uses microenvironment (ME) factors (factors relating the ambient outdoor concentration with the concentration for a specific indoor or vehicular microenvironment) to adjust these concentrations to reflect the conditions in each of 37 microenvironments, including gasoline service stations. Using these factors and exposure pattern data derived from EPA's Consolidated Human Activity Database (CHAD) to assess time spent in each ME for specific population cohorts, the model yielded an estimate of "exposure concentration" for each HAP to which members of the cohort were exposed.

The Science Advisory Board review of NATA criticized the HAPEM version used in the analysis (HAPEM4) for inadequately representing the distribution of exposures (U.S. EPA,EPA-SAB-EC-ADV-02-001, 2001a).[2] In particular, the SAB objected to the use of point estimates for the ME factors. The new version under development, HAPEM5, has several improvements designed to respond to the SAB criticisms. The 37 ME factors can now be input as distributions rather than point estimates, in order to better capture the full distribution of exposures. HAPEM5 can also incorporate spatial variability in air quality estimates within a census tract. Comparison of HAPEM5 with HAPEM4 indicates that mean exposure concentration estimates changed little, but variability was greatly increased in HAPEM5. HAPEM has been used in Houston previously for EPA's recent assessment of local-scale urban air toxics. However the resolution of this model for assessing temporal variability in concentrations of air pollutants is limited, because it is based on average seasonal concentrations. Completion of HAPEM5 is expected in summer 2003.

An alternative exposure modeling option is the newer Air Pollution Exposure (APEX) model. This model has been designed for smaller scale modeling and is based on OAQPS' probabilistic national exposure model for carbon monoxide (pNEM/CO). APEX is part of the inhalation component of EPA's Total Risk Integrated Methodology (TRIM), a time-series multimedia modeling system. APEX can incorporate hourly emission rates and simulate hourly inhalation exposures for all individuals in the sample population, rather than simply using the seasonal average concentration values as HAPEM does. This feature allows for assessment of acute

[2]The SAB comments on HAPEM are included for review as supplementary material for the SAB Council.

as well as chronic exposures, and correlation of exposures with specific activities. APEX also can use a mass balance approach to deriving estimates of concentrations in microenvironments. These features would be advantageous in a local-scale case study such as is planned for Houston, where locations of roads and service stations are likely to impact exposure patterns significantly. However, for this case study APEX would be paired with the less-detailed ISCST3 output, which would not fully utilize the capacity of the model to assess temporal changes in air quality. APEX uses the same data describing human activity patterns (CHAD) as the HAPEM model.

APEX also does not have the capacity to allocate pollution to "source bins" such as point or mobile sources, as HAPEM does, and it has not yet undergone external peer review (release of a beta version is planned for the near future).

At this time, we are proposing to use the HAPEM5 model, once finalized, to evaluate exposures to benzene in the Houston area. We believe it represents a reasonable approach, especially since the revisions to HAPEM5 address the key concerns raised during the SAB review of the NATA study. While the APEX model is promising and may provide the ability for more detailed analysis of exposures in the future, the benefits to the current proposed case study are not expected to be large enough to justify using a model that has undergone less review than HAPEM.

Addressing High-Exposure Subpopulations

To provide a more complete illustration of the effects of reducing benzene exposures to populations in the Houston area, we propose to do supplemental calculations of risk reductions to two high-end exposure groups - service station workers and individuals spending significant amounts of time in homes with attached garages. Both subpopulations spend large portions of their day in microenvironments expected to have above-average concentrations of benzene. Studies of the indoor air concetrations of benzene by EPA and others have found that benzene concentrations in indoor air of homes with attached garages can be two to five times higher than outdoor benzene concentrations. Exposures to service station workers are expected to be high, especially during refueling of vehicles (assuming a full-service station).

We propose to perform these supplemental calculations using the HAPEM5 exposure model. HAPEM includes microenvironmental factors for evaluating exposures at service stations (both indoors and outdoors) and in a residence with an attached garage. We will estimate the size of each of the subpopulations exposed in these environments and their age distribution, and will develop an activity profile for each group to reflect time spent at work or at home each day. We will estimate risk reductions to these groups using the same approach we are proposing for the general Houston population.

Key Benzene Health Endpoints

Our proposed method of assessing benefits from reduction in population exposures to benzene is to estimate the monetary value of the cases of adverse health outcomes avoided and to provide qualitative discussion for non-quantifiable effects likely to occur at ambient concentrations. Effects may be non-quantifiable due to a limited database associating them with benzene exposure or because they are likely to have a threshold concentration above ambient environmental levels.

Cancer

From a dose-response perspective, benzene is a very well-studied chemical with a substantial database of epidemiological data associating it with leukemia. The Integrated Risk Information System (IRIS) entry for benzene identifies the cohort studies of benzene-exposed Pliofilm workers in Ohio (Rinsky et al., 1981, 1987) as the best available data for dose-response evaluation. Due to a lack of historical exposure data, those studies had to rely on assumptions about exposure levels, which have been extensively re-evaluated by other investigators (Crump and Allen, 1984; Paustenbach et al., 1992). IRIS presents a range of unit risk estimates for benzene-induced leukemia (2.2×10^{-6} to 7.8×10^{-6} per $\mu g/m^3$ benzene in air). The ends of the range are derived from estimates reported in Crump (1994) and reflect two alternative approaches to estimating benzene exposures to Pliofilm workers. We note that these maximum likelihood risk estimates do not represent upper bound potency estimates, as is the case with most toxicological data for air toxics; as a result, they are better suited for use in an 812-type analysis where an assessment of typical, not high end, benefits is the goal. We propose to use data from Crump's study (1994) to develop quantitative estimates of avoided cases of leukemia due to implementation of the Clean Air Act Amendments of 1990.

In addition to leukemia, benzene exposure has been associated with other cancers in epidemiological studies, particularly non-Hodgkin's lymphoma (Hayes et al., 1997). However, the data on this endpoint are inconsistent and do not yet support a quantitative evaluation of this endpoint.

Non-Cancer

Benzene has also been associated with a number of non-cancer health effects; however, many of these appear unlikely to occur at levels expected to be found in ambient air (less than 10 parts per billion, based on EPA's NATA study). Benzene exposure at high concentrations has been associated with various hematological abnormalities, including aplastic anemia.

EPA has recently developed a reference concentration (RfC) of 0.03 mg/m^3, based on benzene's hematological effects.[3] The RfC is based on a cross-sectional study by Rothman et al. (1996) of 44 workers in Shanghai, China, who were occupationally exposed to benzene via inhalation. The critical effect on which the RfC is based is "decreased lymphocyte count." The IRIS profile notes that such an effect is a biomarker of exposure, but that the effect itself is of uncertain clinical significance to the average population. The significance of the effect depends both on the magnitude of the decrease in lymphocytes and an individual's baseline lymphocyte level. For example, the effect of reduced lymphocytes might be more significant for individuals whose immune systems were compromised (e.g., those suffering from HIV/AIDS).

At this time, we are not proposing an effort to develop a fully quantitative estimate of non-cancer hematological effects based on the dose-response data underlying the proposed new RfC for benzene. We considered extrapolating the dose-response function based on the data supporting the RfC, in order to estimate "cases" of reduced lymphocyte counts expected at environmental exposure levels. However, the data set supporting the proposed RfC is limited (2 data points) and would not support an extrapolation beyond the benchmark concentration (8.2 mg/m^3) down to the low exposures expected in the environment. We propose therefore, to assess this endpoint by reporting the difference in the number of individuals experiencing benzene concentrations above the RfC under the pre-CAAA and post-CAAA scenarios. While we recognize that exposure above the RfC does not necessarily imply the presence of an adverse effect in a given individual, this estimate nonetheless provides some measure of progress towards reducing the likelihood of adverse hematological effects.

Results from other studies suggest a possible association between benzene and respiratory effects, including reduced lung function, chronic respiratory symptoms, and asthma. However, these studies assessed benzene as a component of volatile organic compounds (VOCs) or engine exhaust and thus could not isolate any effect attributable specifically to benzene (Ware et al., 1993; Laitinen et al., 1994).

Approach to Estimating Avoided Cancer Cases

The goal of this approach is to calculate the expected number of fatal and non-fatal cases of benzene-induced leukemia avoided as a result of the implementation of the 1990 Clean Air Act regulations affecting benzene emissions in the Houston area. We will estimate benefits both on an annual basis for each target year (i.e., 2000 and 2010) and cumulatively across the entire 20-year study period. The approach we are proposing to estimate these benefits is based on the model used to estimate risks due to radon exposure in the National Research Council's BEIR IV report (1988). The approach entails a life table analysis that calculates the probability of contracting (or dying

[3]A reference concentration (RfC) is an estimate, with uncertainty spanning perhaps an order of magnitude, of a daily inhalation exposure of the human population, including sensitive subgroups, that is likely to be without an appreciable risk of deleterious effects during a lifetime.

from) leukemia for a given age cohort in a given time period, conditional on the probability of surviving to that period.

The life table approach allows us to estimate benefits to age-specific cohorts, taking into account age-specific mortality rates, both all-cause and leukemia-specific. This approach also allows us to explicitly integrate into our model an exposure lag parameter, L, that assigns a weight of zero to an individual's last L years of exposure. This approach allows us to estimate a delay in the realization of benefits, but it is not necessarily the same as the "cessation lag" effect previously cited by the SAB (EPA-SAB-EC-01-008, 2001b). The "cessation lag" refers to the estimate of how fast cancer risks in a population will decline to a new steady-state level following a reduction in exposure. The lag, L, represents the period before any benefits begin to be observed. However, given the limited data available on cessation lag, this approach may provide a reasonable first approximation of the effect of latency on benefits (see below.)

We intend to calculate a partial lifetime risk of dying from leukemia, focusing on the study period. We will estimate this risk for both the pre-CAAA and post-CAAA exposure scenarios. The equation we will use for calculating the partial lifetime probability of dying from leukemia (R_0) is:

$$R_0 = \sum_{i=1}^{20} h_i / h_i^* * S_{i-1} * (1 - q_i)$$

where:

R_0 = partial lifetime risk of Leukemia incidence in the study period
h_i = Leukemia mortality rate in the study period i
h_i^* = all-cause mortality rate in the study period i
S_{i-1} = the probability of surviving through period i-1
q_i = the probability of surviving in period i
$(1-q_i)$ = the probability of dying in period i

Data on all-cause mortality rates will be obtained from the United States Department of Health and Human Services' National Center for Health Statistics for years 1990 through 2000 (if available). The estimate of the baseline leukemia mortality rate will be obtained from the National Cancer Institute's SEER database for all available years in the study period. We propose to use mortality data from the latest available year to estimate risks in the latter part of the study period. We will attempt to use Houston-specific or Texas-specific data where available.

The partial lifetime probabilities of Leukemia under the pre-CAAA and post-CAAA exposure scenarios will be estimated for different age subcohorts, assessing risk at five-year intervals using the output data from the exposure model. The cases of Leukemia in each scenario will be estimated by multiplying the probabilities associated with each subcohort by the 2000 census

population for that subcohort, and then summing the results for each target year across subcohorts. (We will also sum results across the entire 20-year study period to generate an estimate of cumulative risk). We estimate the number of leukemia cases avoided as the difference in the number of leukemia cases in the pre- and post-CAAA scenarios.

Survival rates for Leukemia have improved since the time of the Pliofilm cohort, suggesting that a increased percentage of leukemia incidence in 1990-2010 will be non-fatal. Non-fatal leukemia cases represent a separate health endpoint in our benefits analysis. Thus, we plan to estimate benefits using both Leukemia incidence rates and Leukemia mortality rates. The difference between these results will represent the estimate of avoided non-fatal cases of Leukemia.

We will estimate the change in the leukemia mortality rate due to changes in exposure in the pre- and post-CAAA scenarios using a proportional hazards model based on the cumulative exposure multiplicative risk model used by Crump (1994):

$$\Delta h_i = h_i * \beta * \Delta C$$

where:

Δh = the change in the leukemia mortality rate in study period i
h_i = the baseline leukemia mortality rate in study period i
β = an estimate of benzene's carcinogenic potency (risk per ppm-year)
ΔC = the change in cumulative benzene exposure (ppm-years)

The estimate for the beta coefficient will be the maximum likelihood value reported by Crump (1994) for the cumulative exposure linear multiplicative risk model incorporating a five-year exposure lag. (We plan to use a low-end and a high-end beta estimate, based on different assumptions about the exposure of the Pliofilm workers, to generate a range of benefit estimates; see below.) Crump also estimated coefficients for this model assuming a three and zero year lag; however he reported that the five-year lag assumption combined with the multiplicative risk model produced the best fit to the data. The true latency period for benzene-induced leukemia (and hence the corresponding cessation lag period for the full benefits of exposure reduction to be realized) is uncertain, however, and alternative assumptions about the lag structure could also be reasonable.

The estimates of the change in benzene exposure for the target years 2000 and 2010 will be derived from the exposure model output for each age cohort. We will need to interpolate estimates of exposure concentrations for years in between the target years. Our initial proposal is to perform a linear interpolation of concentration changes between the target years.

Some assumptions inherent in these calculations are that Crump's exposure-response modeling results for the epidemiology study (Pliofilm cohort) can be applied to the general population and that the relative risk model obtained applies to all age groups. The applicability to

the general population is a source of uncertainty, but the same assumption was also inherent in the cancer potency estimates already adopted by EPA. The assumption of applicability to all age groups is generally a reasonable one and is commonly used (this assumption is also apparently integral to Crump's analysis). To the extent that the cause(s) and pathogenesis of some childhood leukemias may be different from those of adult leukemias, the inclusion of the childhood leukemia rates may overestimate benefits to the younger subcohorts. However, these younger subcohorts may be more sensitive to benzene exposure, or benzene exposure may contribute similarly to the development of childhood leukemias; thus, it seems reasonable and prudent to include them.

Cessation Lag

EPA's Science Advisory Board has defined "cessation lag" as the period it takes for risk to decline to a steady state level following a reduction in exposure.[4] For most, if not all, health effects associated with air toxics, there will be little or no data estimating the length of this period. Therefore, in order to develop a reasonable temporal stream of benefits, we must rely on available data that attempt to characterize the disease *latency* (the time between a critical exposure and the development of symptomatic disease or death).

Crump (1994) evaluated benzene risk using several models based on data from the Pliofilm cohort. His cumulative exposure models employ a "lag", L, that assign a weight of zero to the last L years of an individual's exposure. This model assumes that exposures during the most recent L years do not affect the mortality rate. Crump tested lags of 0, 3, and 5 years and found that a lag of 5 years produced a significantly better fit to the data than lags of 0 or 3 years. These findings would suggest that the *latency* period for benzene-induced leukemia is *at least* five years, but could be more. It also implies that zero benefits would accrue in the first five years following an exposure change.

Also, a recent paper by Silver et al. (2002) that evaluated the effect of follow-up time on risk estimates in the Pliofilm cohort found that the relative risk of leukemia peaks in the first few years following cessation of benzene exposure and that exposures five to ten years prior to the cutoff of exposure have maximal impact on risk. Together with Crump's findings, this suggests that a new steady state risk level may not be reached before at least five years and possibly 10 years following an exposure reduction.

We are proposing using an exposure lag of 5 years in the HAP case study when estimating the time stream of benefits due to reductions in benzene exposure, as a first approximation to the "cessation lag". However, we acknowledge that the database regarding the latency of benzene-induced leukemia, on which we must base our framework, is quite limited and uncertainty in the mode-of-action of benzene carcinogenesis makes it difficult to assess the biological plausibility of the values reported in these studies. As a result, we propose to evaluate the effect of alternative lag

[4] U.S. EPA, *Arsenic Rule Benefits Analysis: An SAB Review*. August 9, 2001. EPA-SAB-RSAC-01-005.

structures (e.g., zero years, ten years, or five-years with a "phasing-in" of benefits) on benefits as part of a sensitivity analysis.

ECONOMIC VALUATION OF EFFECTS

This section describes our approach to assigning economic value to the estimated benefits of reductions in ambient benzene concentrations. The scope of the valuation methodology is determined by the prior steps in the case study, which necessarily limits monetization to those health effects for which concentration-response estimates are available. This is not meant to imply that the ecological and non-quantified health benefits of benzene reductions have no value, only that within the framework of this case study we are unable to estimate that value.

Overview of Approach

We plan to apply valuation methods that are consistent with those employed to value the benefits of the Second Prospective analysis of criteria pollutants. For example, the valuation of fatal cancers will rely primarily on the base value of statistical life (VSL) estimates used for PM mortality valuation. In the benzene exposure case, however, there is the additional consideration of a potential "cancer premium" that many analysts believe to be an aspect of the health risk context that is important for valuation. In addition, the valuation of non-fatal cancer cases is not reflected in the criteria pollutant analysis. Finally, there is the consideration of non-cancer health effects associated with benzene. Although no quantification of non-cancer effects is planned for the case study, we plan to provide some economic context for these real benefits of benzene control programs by providing, where possible, cost-of-illness estimates and a summary of potentially relevant willingness-to-pay values for the critical effect of concern (decreased lymphocyte count).

For non-fatal cancer case valuation we propose to follow recent SAB advice on this topic given during a consultation in 2001 regarding a possible arsenic rule-making by EPA's Office of Water (EPA/SAB 2001b). Those recommendations have not been implemented by EPA to date, in part because the arsenic drinking water rule was finalized based on a prior analysis, but we believe the recommendations are relevant here, with some adjustment as outlined below.

Valuation of Cancer Endpoints

Fatal Cancers

Fatal cancers will be valued on a per-case basis using the VSL estimate developed from meta-analysis of estimates in the relevant economic literature. The approach to developing this VSL estimate is described in depth in Chapter 8 of the Analytical Blueprint, Economic Valuation. The estimate developed from the meta-analysis described there reflects valuation of immediate, non-

cancer risks. As a result, this value needs to be adjusted to reflect the timing of the manifestation of the risk (addressed in a separate section below), and the potential for a "cancer premium."

The potential for a cancer premium was explicitly acknowledged in the previously cited SAB report, and derives from the observation that cancer victims may suffer greater fear or dread than the victims of the causes of death involved in VSL studies that underlie the meta-analysis used here (see page 17 of their report). If health individuals perceive that a death from cancer is worse than a death from another cause, then it is plausible to conclude that they would be willing to pay more to avoid that type of death. The SAB concluded that there was little reliable information on how large the premium might be, however.

The SAB did nonetheless endorse "the addition of estimates of the medical costs of treatment and/or amelioration for fatal cancers to the VSL as a lower bound on the true value of avoiding fatal cancers." In our case, these estimates would relate to the treatment costs for a fatal case of leukemia. EPA is aware of no careful, comprehensive estimates of the cost of illness for leukemia treatment, and leukemia is not one of the cancers currently covered by EPA's Cost of Illness Handbook, but costs for other, potentially similar cancers may be appropriate for this purpose.[5] Resource limitations preclude the development of a new primary cost-of-illness estimate to support this study, but EPA plans to conduct a review of the health economics literature to ensure that the best available estimates are used.

Non-fatal Cancers

Estimates of the value of avoiding non-fatal cancers are sparse in the economic literature. The SAB arsenic panel, commenting on a valuation strategy for non-fatal bladder cancer, recommended the use of two estimates that could be interpreted as the "two extreme estimates available in the literature" as bounds in an uncertainty analysis. The two estimates are for the value of avoiding chronic bronchitis obtained by Viscusi, Magat, and Huber (1991), and the value of avoiding nonfatal lymphoma obtained by Magat, Viscusi, and Huber (1996). Both estimates are willingness to pay estimates, but both are derived from mall intercept studies that raise concerns about the representativeness of the sample. Chronic bronchitis is a serious chronic condition that the EPA Office of Drinking Water has interpreted to be similar in severity to nonfatal cancer.

We plan to follow the SAB's advice for valuation of nonfatal cancers, but to use a chronic bronchitis value consistent with that used in the Second Prospective criteria pollutant analysis, which incorporates downward adjustments in severity of the chronic bronchitis case that are consistent with the type of case usually associated with air pollution exposure.

[5] See **http://www.epa.gov/oppt/coi/** for access to EPA's Cost of Illness Handbook.

Consideration of Cessation Lag

As discussed in prior sections of this chapter, reduction in exposure to benzene leads to reduction in cancer cases after a period of cessation lag. In economic terms, it is plausible to assume that individuals would prefer avoidance of immediate health effects relative to avoidance of health effects with a delay, suggesting that their willingness to pay to avoid delayed health effects is affected. Because the underlying VSL estimates are largely for immediately manifest risks of death, the VSL estimate needs to be adjusted to account for the effect of the cessation lag on willingness to pay.

We plan to make this adjustment by discounting the VSL estimate by the period of cessation lag using two alternative discount rates consistent with those applied in the Second Prospective as a whole (i.e., a primary estimate using a discount rate of 3 percent, and an alternative estimate using a discount rate of 7 percent).

UNCERTAINTY ANALYSIS

This section discusses of proposed efforts to characterize uncertainty and variability in the benefits estimates for the benzene analysis.

Emissions and Air Quality Modeling

The uncertainties associated with these two elements of the analytical chain are complex, and we currently anticipate that resource limitations will preclude a probabilistic, quantitative treatment of the effect of these uncertainties on the benefit results. Therefore, at this time, we propose to address uncertainties in these elements qualitatively, by identifying the key uncertainties, assessing their relative magnitude (e.g., major versus minor) and their likely impact on our results.

Exposure

The HAPEM model incorporates variability and uncertainty distributions into its exposure modeling algorithm, facilitating the characterization of variability and uncertainty in exposure. Among the stochastic elements in the HAPEM model are variability in demographic characteristics, activity patterns across demographic groups (e.g., time spent in different microenvironments), and variability in work location. The version of HAPEM currently being developed (HAPEM5) also incorporates variability and uncertainty in microenvironment factors that relate concentrations in a microenvironment to ambient levels, and spatial variability in ambient HAP concentrations within census tracts. The output of the HAPEM model will provide distributions of exposure concentrations for different demographic groups that can serve as inputs to a probabilistic Monte Carlo analysis of the benefits of reductions in benzene exposure.

Dose Response

The major sources of uncertainty in this part of the analysis center on uncertainty surrounding the true value of the beta coefficient describing the carcinogenic potency of benzene and the true shape of the concentration response function at the lower concentrations expected to be found in ambient air.

Uncertainty in the Concentration-Response (C-R) Coefficient (Beta)

Much of the uncertainty surrounding the carcinogenic potency estimates for benzene arises from uncertainty in reconstructing the exposures of the Pliofilm workers. To reflect this uncertainty, we propose to calculate the primary benefit estimate of the reduced risk of benzene-induced leukemia as a range of values. This lower end of this range will be calculated using the beta value that is associated with the lower Paustenbach exposure estimates in a multiplicative cumulative exposure risk model with L = 5 years (1.1E-02); the upper end risk reduction will be calculated using the beta value that is associated with the higher Allen exposure estimates and a multiplicative cumulative exposure risk model with L = 5 years (1.7E-02). Because IRIS does not assign probabilities to the potency estimates calculated using alternative exposure assumptions, we do not assign probabilities to the alternative benefit values calculated using those beta values. Thus, the range should not be interpreted as a statistical confidence interval; the primary benefit estimate is expected to fall within the reported range of values, however.

We will also estimate an uncertainty distribution around each of the beta values used to calculate primary benefits. This distribution will capture the uncertainty in the measurement of the beta value, separate from uncertainty in the exposure reconstruction. For each beta value, we will use the the reported estimate in the study as the best estimate of the mean of the distribution of C-R coefficients. We will then characterize the uncertainty surrounding the estimate of the mean C-R coefficient as a normal distribution, with a standard deviation derived from the standard error of the reported beta value. These distributions can then be used as inputs into a Monte Carlo analysis of benefits that would generate a distribution of benefits results for each of the two ends of the benefits range.

Uncertainty in the Dose/Response Model

The mode of action for benzene-induced leukemia is complex, and despite significant advances in our understanding of the process, much remains uncertain. As a result, the true shape of the dose response function can not be known with certainty. EPA has concluded that there is insufficient evidence at present to reject a linear dose-response curve for benzene, and thus recommends use of the low-dose linear model.

However, there is some evidence suggestive of a non-linear dose response at low doses, and risk estimates would be significantly affected if a non-linear model were to be adopted. Ideally, EPA would address this model uncertainty in the proposed benzene case study using a sensitivity

analysis that illustrates the effect on benefits of assuming one or more alternative dose-response shapes (e.g., a supralinear and a sublinear model). Identifying suitable alternative functions from the many non-linear possibilities appears to be a quite difficult task, however, due to the lack of observed data in the low-dose range and the remaining uncertainties surrounding the benzene mode of action. Therefore, we are not proposing to recommend a quantitative evaluation of dose-response model uncertainty but will instead include a qualitative discussion of its possible impact on benefits.

Valuation

Uncertainty analysis for the valuation component will largely depend on analytic choices made in the criteria pollutant analysis. Uncertainty in the base VSL estimate used for fatal cancer will be characterized based on the Kochi et al. (2003) results presented in Appendix H of this document. The project team continues to explore options for characterizing uncertainty in the medical cost of treatment component of fatal cancer valuation. One option is to rely on estimates of measurement error and/or variability in cost of illness as it is currently estimated based a national survey method. The approach for non-fatal cancer valuation that we propose implies uncertainty characterized by a uniform distribution of values within the bounds of the "two extreme estimates available in the literature" for chronic bronchitis.

In the cases of fatal and non-fatal cancers, these characterizations of uncertainty are appropriate for inclusion in a probabilistic framework. Uncertainty in valuation of the effect of a cessation lag, however, is more appropriately addressed by a sensitivity test. We propose to evaluate the effect of using a seven percent discount rate rather than the three percent rate we plan to use for the primary analysis.

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